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ORIGINAL MEMOIRS.

AN ANALYTICAL AND STATISTICAL REVIEW OF ONE THOUSAND CASES OF HEAD INJURY.

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THE analysis of so large a number of complicated cases of head injury is necessarily prolix, but can hardly fail to have interest and value to those who study cranial and intracranial injuries. It will serve as a supplement to an earlier work ("Traumatic Injuries of the Brain and its Membranes," Phelps, 1897–1902), and will practically corroborate the general statements made at that time.

The cases cited are limited now, as they were then, to those personally observed; and all inferences, conclusions, and statements of fact are based exclusively upon that observation. Extracranial injuries other than fractures have not been noted, as with the advent of aseptic precautions they have ceased to be of practical importance except as aids in the diagnosis of intracranial lesions. Cutaneous wounds which through neglect have become infected are still readily amenable to local antiseptic treatment, even in the exceptional instances in which they are responsible for secondary intracranial inflammations.

Head injuries, as they will be considered, are most conveniently classified primarily as *fractures of the cranial vault,* *fractures of the cranial base,* and *independent injuries of the cranial contents.* They most frequently complicate each other, and possibly in a majority of cases all these conditions are present together; but any one may exist practically by itself, and its separate existence may be recognizable and may determine prognosis and treatment.

SUMMARY OF CASES.

	Cases	Recoveries	Deaths	Necropsies
Fractures of cranial base....	570	259	311	203
Fractures of cranial vault...	213	152	61	46
Independent injuries without demonstrable fracture	217	130	87	63
Totals..................	1000	541	459	312

The class last mentioned includes cases in which the absence of fracture is assumed from the absence of characteristic symptoms; and as in many instances a fractured base has been discovered post mortem when no indications had existed during life, it doubtless includes a very considerable number of cases which were really cases of fracture.

It is perhaps the most notable feature of this table that the recoveries exceed the deaths in number; and that even with fractures that involve the cranial base, which were long supposed to be almost if not quite invariably fatal, the recoveries so nearly approximate the fatalities.

FRACTURES OF THE SKULL.—These may be as variously classified in accordance with their character and location as are those of the extremities; but their simple division into fractures of the vault and fractures of the base is distinctive and therefore the most convenient. Each of these grand divisions may be subdivided into simple, compound, depressed, punctured, and complicated, as are other fractures, and occur with varying degrees of frequency and importance as they affect one region or another.

It has been found in this collection of cases that the vast majority of fractures of the base originate in the vault, and are continuations of fissures, which, beginning at the point upon which the impact of external violence was received, follow the line of least resistance into the corresponding basal fossa, where they terminate, or whence they extend into the adjacent or even into all the fossæ. They should be considered as fractures of the base rather than of the vault because it is from implication of that region that their characteristic conditions depend. Various theories explanatory of the extension of fractures from the vault to one region of the base rather than to another were noted in that earlier work (*lib. cit.*) to which reference has been made, and to which it will often be necessary to refer hereafter. The larger experience has suggested no modification of what was then said, and its repetition may be avoided as not essential to the present purpose.

Direct Fractures.—The whole number of cases in which with force transmitted from the vault the fracture was confined to the base, as verified by subsequent necropsy, was forty-six. In seven of these there was coincident but independent fracture of the vault, which sometimes involved the base in some distant region. It is not impossible that there were unrecognized direct fractures among cases which terminated in recovery, or in death without post-mortem examination, since of those discovered many did not contribute to the fatal result, and fully one-half were unaccompanied by symptoms indicative of fracture.

There were additionally cases of fracture of the base from force directly applied. These include eighteen of pistol-shot origin, a small number of perforations of the orbit with punctured wound, and three or possibly four in which orbital or ethmoid fracture was produced by great violence inflicted in the orbital or nasal region.

No history was obtainable in one-third of the cases in which fracture was limited to the base; in one-half the number the patients had fallen variable distances, as from the mizzen top into the hold of a vessel to a mere fall on the street or to the floor from a bench in a police station; in the remaining cases they were struck upon the head or knocked down by some moving vehicle; and in two exceptional instances the head was forcibly flexed in the occipitomental diameter upon an unyielding obstruction by a descending elevator.

Any one or more of the basal fossæ may be involved in a direct fracture, or it may be limited to the petrous portion. All the fossæ were included in two cases, and all but one anterior fossa in three others. The middle fossa was alone fractured in twenty cases, and was implicated in thirteen more. A single orbital plate, or the petrous portion, was sometimes the only part to be involved. So far as it was possible to determine the point or region upon which the force of impact had been exerted, its relation to the site of fracture was inconstant. It is not as obvious that the direct basal fracture was produced in the line of least resistance, as it seemingly was in the cases where fracture was prolonged from a point of impact upon the vault. In some instances in which a fracture limited to an orbital plate or to a sphenoid process followed a blow upon the eye, force must have been transmitted directly through the soft parts. In the larger number of cases fracture was produced indifferently upon the side of injury, or upon its opposite, or in any of the cranial diameters. In such case it becomes difficult to determine whether transmission was through the bone or through the cranial contents, as well as clinically unimportant. There were two instances in which force was transmitted from the feet through the body; but in one, which was fatal, there was no fracture.[1] The other, terminating in recovery, is of sufficient interest to quote in brief.

CASE I.—A man fell nine stories to the ground, striking upon his feet, his fall having been broken by several pieces of projecting timber. He was found sitting upon the ground, conscious, rational, and complaining of severe pain in his head; temperature 98.8°; pulse 60; respiration 15; free hemorrhage from his right ear. On admission to the hospital his general symptoms were unchanged. He was subsequently restless, irritable, and delirious, with temperature rising to 103° on the second day. Oozing from the ear continued till the fourth, and delirium till the sixth day. Vesical and rectal control was retained.

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[1] Phelps: Traumatic Injuries of the Brain and Its Membranes, Case CXCVI.

The intracranial complications which attend direct basal fractures do not differ from those which may occur when the fracture has its beginning in the vault, and like them they are usually concomitant rather than resultant. The exceptional instances were those in which the middle meningeal artery or a basal sinus was ruptured; and two in which a frontal lobe was lacerated by fragments of an orbital plate.

The subjoined cases are illustrative of this form of fracture.

Case II.—Patient knocked down by a bicycle; right eye closed by hæmatoma; abrasions of head and face; comminution of corresponding orbital plate, with fissures; one fissure running upward into frontal bone, and two backward and respectively through opposite clinoid process and through corresponding greater sphenoid wing into petrous portion and finally into foramen lacerum posterius.

Case III.—No history obtainable; contusion of both eyes; comminuted fracture of one orbital plate with displacement of fragments and fine fissures; one fissure ran through ethmoid and sphenoid bodies, one through middle fossa and along anterior petrous surface, and one through optic foramen.

Case IV.—No history obtainable; hæmatoma of right orbit; transverse fracture of corresponding orbital plate.

Case V.—Patient fell in the street. No external injury; separation of greater wing of sphenoid from squamous portion of temporal bone.

Case VI.—Patient fell ten or fifteen feet through a hatchway; fissure through internal auditory meatus and into tip of greater wing of sphenoid bone.

Case VII.—No history obtainable; wound in right occipital region; linear fracture running transversely across middle of right petrous portion.

Case VIII.—No history obtainable; wound over occipital tuberosity; fine fissure confined to posterior petrous border.

Case IX.—Patient jumped from fifth story window; two independent fissures, each limited to a petrous portion.

Case X.—Patient fell one story; struck upon left side of head and body; scalp wound in left temporal region; fracture extending from outer part of anterior surface of right petrous portion through body of the sphenoid bone into its lesser wing.

Case XI.—No history obtainable; occipital wound; fracture beginning at foramen magnum, and extending upward into opposite inferior posterior fossa.

Case XII.—No history obtainable; fracture extending from anterior petrous surface to foramen spinosum, lacerating arteria meningea media.

Case XIII.—Patient fell from his seat in a police station; two fractures of the vault, and one limited to the base running along whole length of anterior petrous surface.

Case XIV.—Patient fell down one flight of stairs; fracture of both orbital plates, left greater wing of sphenoid bone, left petrous portion, and basilar process of occipital bone.

Case XV.—Patient fell from the mizzen top into the hold of a vessel; fractures confined to the base; six fissures involving all the basal fossæ; one primary fissure began at the foramen magnum and formed five subdivisions, and a sixth was independent and confined to the crista galli and sella turcica.

Case XVI.—Patient was struck upon the occiput by a descending elevator and his head forced forward upon a railing; fracture confined to anterior fossæ, and extending from posterior border of cribriform plate by a wide curve forward and outward, and then inward through both orbital plates to a corresponding point on the left side. The roof of each orbit was elevated and tilted forward, and the frontal sinuses were made continuous with the cranial cavity. The right frontal lobe was extensively lacerated by an orbital fragment.

SYMPTOMS AND DIAGNOSIS.

The symptomatology of cranial fracture has been much confused by a failure to distinguish the symptoms of the fracture itself from those of the intracranial complications which so frequently accompany it, and give to it a factitious importance. The abnormalities of pulse, temperature, and pupils—the loss of consciousness and other morbid conditions—which are still enumerated as symptoms of fracture, indicate intracranial lesions which are only in rare instances even dependent upon the fracture which they accompany. They equally occur when no cranial injury has been sustained, and are then manifested by the same general indications. They result from a common cause, but are essentially independent of each other. It is only occasionally that an osseous fragment may rupture an important vessel or lacerate the cerebral substance.

The distinctions between symptoms of a fracture and those of the concomitant cerebral or meningeal lesions have more than an academic interest, since a well-defined conception of the significance of symptoms is essential to correctness of prognosis and treatment.

The direct symptoms are few, and not difficult to discover. Fractures confined to the vault are to be considered apart from those which primarily or secondarily involve the base. They are to be recognized by tactile or by visual sense, which are always practicable methods; and there is no justification for the neglect to resort to both when one is insufficient for exact diagnosis. If the fracture be compound there can be no doubt of its existence, provided the wound be of sufficient size to disclose the osseous surface. If the wound be too small for thorough exploration the fracture may be regarded as essentially of the simple variety. The simple fracture if depressed may often be recognized by palpation through the layers of the scalp; but if doubt exists, or if from symptoms of intracranial complication suspicion arises, certainty should be attained by incision and direct inspection. This covers the whole ground of diagnosis—tactile or visual examination—and if necessary to that purpose sufficient and unhesitating incision down to the cranial surface. It may happen that a fracture confined to the vault, or extended to the base, affords no local indication of injury having been sustained—neither wound, hæmatoma, nor contusion. There is then, in the absence of intracranial symptoms, no warrant for exploration; and it is fair to assume that if fracture exist it is immaterial.

The diagnosis of fractured base is sometimes incidentally made in the course of an exploration of the vault by establishment of the continuity of a fissure from one region into the other; this, however, is exceptional and likely to be confined to cases in which the vault is extensively comminuted, or in which search is being made for the source of an epidural hemorrhage.

Hemorrhage.—The one indication of basal fracture, and the only one upon which dependence can be placed in the great majority of cases, is a cranial or intracranial hemorrhage which through some channel becomes visible upon or beneath the surface. Its source may be the vessels of the diplöe, the meninges, or the brain; and its escape may be from the ear, nose, or mouth, or into the subconjunctival or subcutaneous tissue.

The absolute and comparative frequency of these several forms of hemorrhage is indicated in the following table.

		Recovered	Died	Total	Necropsies
Hemorrhage from the ear...		166	119	285	69
Hemorrhage from nose and mouth..................		39	51	90	32
Subconjunctival hemorrhage.		9	8	17	7
Subcutaneous hemorrhage	mastoid	3	6	13	9
	cervical......	0	3		
	orbital.......	0	1		
				405	

The hemorrhage from the ear is not only the most frequent, but also is the one of paramount importance. As a positive sign it may be considered pathognomonic of fracture of the petrous portion of the temporal bone involving the internal auditory passage and followed by rupture of the tympanum from pressure of extravasated blood. The single exception to this as an absolute rule was encountered in a case of gunshot wound inflicted at short range immediately in front of the ear, in which a slight hemorrhage was occasioned by rupture of the tympanum from concussion. In every other instance in which ruptured tympanum and hemorrhage occurred, post-mortem examination showed a petrous fracture through the auditory canal, and in no instance in which hemorrhage from the ear had been absent was such a fracture disclosed. If a petrous fracture did not involve the internal auditory canal it would, of course, afford no outlet for the blood, which may come from either the osteal or meningeal or even from the cerebral vessels. The large number of cases observed would seem to warrant the conclusion that no extremity of violence otherwise inflicted upon the cranium can rupture the tympanum by concussion; and that the instances of such rupture from atmospheric disturbance or bullet impact are very exceptional.

In making this diagnosis care must be exercised in examining the external auditory meatus, as the cranial or intracranial hemorrhage is occasionally simulated by a hemorrhage into that passage from an external wound. There were nine such exceptional cases in the present series, in four of which the absence of fracture was further verified post mortem. In each instance blood had trickled into the meatus from some wound of the scalp or external ear, or had issued from some wound within the meatus itself; and in each it had been mistaken on primary and cursory examination for the usual indication of fracture. The necessity for this precautionary examination of the external parts for the source of an aural hemorrhage is so apparent that reference to it would seem supererogatory were it not that it is so generally neglected. The wound in the tympanum is not often visible after the hemorrhage has ceased because it is usually simple, and primary union is the rule; but the absence of any possible external source of hemorrhage is quite sufficient for diagnosis. The flow may be trivial or profuse; it may cease at once or be prolonged for days; but neither the quantity nor the duration of the flow is material.

The nasal and subconjunctival hemorrhages though often noted are much less frequently indicative of fracture. Their dependence upon local injury in many cases is evident, and their intracranial origin in many others is scarcely less open to question; but their significance in perhaps a majority of cases is purely conjectural. It follows that their frequency as symptoms of cranial fracture cannot be stated with the positive certainty which was possible in cases of hemorrhage from the ear, and that their number as stated in the preceding tabulation must be regarded as only approximate.

If a subconjunctival hemorrhage appears at once, together with an orbital or palpebral ecchymosis, it is probably a part of the general local contusion; but if it appears only after an interval of several hours, or of one or more days, and without subcutaneous extravasation, it is fair to assume a fracture involving some part of the orbital wall. If the hemorrhage from the nose or mouth is profuse and there is no local lesion or history of injury to the face, it is probably the result of fracture through the anterior or middle fossæ. On the other hand, the beginning of a slow oozing from the nose after the lapse of twenty-four hours has a similar significance. Hæmatemesis in head injuries, in the absence of other explanation, always suggests cranial fracture, partly as an evidence of the profusion of nasal hemorrhage, and in part as a possible result of fracture through the osseous wall of the pharynx. The concurrence of symptoms of intracranial complication will confirm the existence of a fracture which the nature of the hemorrhage has rendered probable.

The cases of nasal and subconjunctival hemorrhage tabulated were in each instance uncomplicated by other hemorrhages. In addition somewhat more than 40 per cent. of hemorrhages from the ear were accompanied by one or the other or by both of these, but usually by the one from the nose. The subsidiary hemorrhages, as they may be termed, then ceased to be of diagnostic importance, except as indicating the possible extent of the fracture.

The subcutaneous hemorrhages of cranial or deeper origin are usually manifest in the mastoid region, and, together with the exceptional instances occurring in the posterior cervical region, are with proper limitation of observance as pathognomonic as are hemorrhages from the ear. There were, in addition to the nine cases in which there was no other than mastoid hemorrhage, thirteen in which it was complicated by hemorrhage from the ear, and two by hemorrhage from the nose. Seven of these twenty-four cases, of which five were simple mastoid hemorrhages, afforded opportunity for post-mortem examination; the mastoid ecchymosis was observed primarily in all, and was found post mortem to be continuous with a large extravasation into the occipital region of the scalp. The fracture in each traversed an inferior occipital fossa, and in five involved the groove for the lateral sinus. In other cases, including most of the recoveries, the mastoid ecchymosis, which was sometimes accompanied by œdema, did not appear till later—from the second to the sixth day—and in succession to a hemorrhage from the ear. These would seem to be cases of fissure extending from the petrous portion into the mastoid process.

It is possible that blood from a temporal or temporoparietal hæmatoma may gravitate into the mastoid area; but sufficiently careful examination will serve to exclude this source of error. Such a possibility having been excluded, a mastoid ecchymosis may be regarded as an unfailing indication of fracture, the site and extent of which may be inferred from attendant conditions.

An œdema of the mastoid region without hemorrhage was noted in only two instances. One of these was cited in the earlier series,[2] in which a fracture traversed the groove for the lateral sinus, and was accompanied by obstruction of that vessel by thrombosis. The occurrence of this symptom will necessitate the joint condition of a venous obstruction to cause the œdema and of a fracture to permit its appearance in a cranial region.

In some instances the mastoid extravasation extended into the neck, and occasionally a subcutaneous hemorrhage was limited to that region; in either event the hemorrhage followed extensive occipital fracture. The earlier and the more considerable the visible hemorrhage, the greater the probability of the existence of cranial comminution or of open fissures. A simple closed fissure would rarely be attended by external hemorrhage sufficiently large to become subcutaneous, and then only after the lapse of some days.

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[2] *Lib. cit.*, Case LXII.

The mastoid and cervical are perhaps the only subcutaneous hemorrhages likely to be brought in question as indicating basal fracture. In a single instance perceptible orbital hemorrhage resulted wholly from comminution of an orbital plate. Punctured fractures when they occurred in this cavity were usually attended by such hemorrhage, but it was the result of the wound of the soft parts rather than of the bone. A hemorrhage into the zygomatic fossa from fracture of the greater wing of the sphenoid bone is conceivable, but if recognized would hardly be distinguishable as such.

Symptoms of basal fractures, aside from hemorrhage, are few and of exceptional occurrence. They are:

Serous Discharges;
Extrusion of Brain Tissue;
Implication of Cranial Nerves;
Localized Pain.

Serous Discharges.—These were observed in twenty-six cases, nine of which proved fatal, with nine necropsies. They were all from the ear with the exception of one from the vault, and one from the nose. In one instance the discharge was from the pharynx as well as from the ear. In a certain number of cases the source of the discharge may be considered uncertain; in the remainder all the sources to which it has been ascribed were exemplified. The fluid itself may be the serum of blood extravasated within the cranium, the cerebrospinal fluid, the serous effusion of an arachnitis, or the viscid discharge of a middle-ear inflammation. The determination of its nature must be based upon the period of its occurrence, its relation to hemorrhage, its chemical and physical properties, and the significance of concomitant symptoms. The chemical characters of the discharge were not given much attention as they were usually unimportant in diagnosis and the discharge difficult to obtain in sufficient quantity for examination.

There was primary hemorrhage in twenty-one of these cases, of which seven were fatal. The hemorrhage in seventeen cases preceded the discharge by an interval of from a few moments to several (6) days; and in four they were synchronous. In all such cases the serous discharge has no importance in diagnosis, as the certainty of fracture is already assured by the fact of hemorrhage. In one of the five cases in which there was no hemorrhage from the ear there was primary mastoid ecchymosis and œdema.

An analysis of the twenty-six cases shows that in four the discharge was accompanied by other undoubted symptoms of aural inflammation; that in four it was the result of a declining stage of inflammation; and that in fourteen it was the cerebrospinal fluid, as it probably was in four others in which there was more or less reason to question its exact character. The recognition of cerebrospinal fluid was based in all the recovering cases upon profusion, limpidity, and immediate occurrence; and in cases followed by necropsy upon the additional evidence of positive and negative conditions—the presence of fracture and absence of simple or infected subarachnoid serous effusion, and the absence of notable epidural or subdural hemorrhage.

Extracts from the histories of some of the cases of serous discharge from the ear will serve to illustrate the several conditions under which it occurs.

CASE XVII.—*Serous Discharge from an Inflamed Ear; Necropsy.*—The patient fell down stairs; slight primary hemorrhage from the right ear and subconjunctival hemorrhage on the second day; progressive stupor, and on the eighth day unconsciousness, with considerable serous discharge from the ear, which became purulent on the day following; temperature on admission 100.8°, and then normal until the twelfth and last day of life, when it rose to 101.8°.

Lesions.—Limited areas of meningeal contusion, each 1 in. by 1½ in. in its diameters, just posterior to fissure of Rolando; minute opening in posterior surface of right petrous portion through which pus exuded; large subarachnoid serous effusion in right posterior basal fossa; suppuration and disintegration of mastoid cells.

Case XVIII.—*Serous Discharge, Probably from an Inflamed Ear; Recovery.*—The patient fell from a ladder and struck upon his head; semiconscious with vertigo on his admission to the hospital; mastoid swelling and profuse hemorrhage from the right ear, which continued till the third day; oozing from the ear on the fifth, which ceased on the sixth, and recurred on the seventh day; previous delirium increased; severe pain in the ear, which was continuous up to the day of his discharge from the hospital on the fourteenth day.

Case XIX.—*Serous Discharge from the Ear in a Declining Stage of Hemorrhage; Recovery.*—The patient fell from a second floor window; coma and profuse hemorrhage from the mouth and left ear; followed by hæmatemesis; hemorrhage from the ear continued thirty-six hours and was succeeded by a gradually diminishing discharge of bloody serum.

Case XX.—*Discharge of Cerebrospinal Fluid from the Ear; Recovery.*—The patient, ten years of age, fell 40 feet; transient loss of consciousness; synchronous hemorrhage and watery discharge from the right ear; watery discharge continued till the fourth day; temperature normal; no general symptoms.

Case XXI.—*Late Discharge of Cerebrospinal Fluid from the Ear; Necropsy.*—Primary hemorrhage from nose and both ears, and later profuse hemorrhage from the pharynx; moderate discharge of watery fluid from the left ear on the fourth day.

Lesions.—Fracture through both middle basal fossæ; comminution of left petrous portion; extensive pial hemorrhage on the right side; marked cerebral œdema; no subarachnoid effusion, but convolutions of the left hemisphere flattened.

Case XXII.—*Discharge of Cerebrospinal Fluid from Both Ears; Recovery.*—The patient, three years of age, was knocked down by a street railway car; no hemorrhage; primary free flow of a watery fluid of a bluish tint, without tinge of blood or viscidity; ceased in twenty-four hours; no general symptoms; temperature rose to 104° and then gradually receded.

Case XXIII.—*Late Seropurulent Discharge from the Ear, Probably a Subarachnoid Inflammatory Effusion; Necropsy.*—The patient was thrown from his truck and struck upon his head; free hemorrhage from right ear with mastoid ecchymosis, followed at once by severe frontal headache and high temperature; hemorrhage continued till third day, becoming more and more serous in character; discharge seropurulent on the fourth day; death on the sixth day, having had marked symptoms of arachnitis.

Lesions.—Fracture extending from occipital region along groove for lateral sinus, and through mastoid process, into petrous portion; pial hemorrhage in right occipital fossa; small hemorrhages into substance of pia mater, each of the size of a pea, in the right frontal region; and pial vessels much distended over the whole vertex; subarachnoid seropurulent effusion over right hemisphere; lateral ventricles filled with a brownish fluid.

CASE XXIV.—*Discharge of Cerebrospinal Fluid from the Nose; Recovery.*—The patient was found unconscious in the street with profuse hemorrhage from mouth and nose, much orbital ecchymosis, and a fractured nasal process of the superior maxillary bone; copious sanguinolent discharge from the nose on the eighteenth day, which began to diminish three days later and ceased only on his discharge from the hospital at the end of another week. The fracture was found to extend into the ethmoid plate.

CASE XXV.—*Discharge of Cerebrospinal Fluid and Later of Subarachnoid Inflammatory Effusion from Ear and Pharynx; Necropsy.*—The patient was knocked down by a bicycle and struck his head upon the pavement; could stand up and walk. On admission to the hospital: Slight hemorrhage, with a synchronous escape of considerable serous fluid from the right ear; back of pharynx blood-stained; no general symptoms. Second day: Constant expectoration of straw-colored fluid; free and constant discharge of watery fluid from the ear, which continued till his death on the fifth day. The patient became restless on the second day, with retching and vomiting; and on the fourth day delirious with twitching of the muscles of the extremities and exaggerated reflexes, which continued till his death. Temperature rose to 104.6°.

Lesions.—Fissure began at the left clinoid process, ran forward through the ethmoid bone, comminuted the right orbital plate, and then divided into two branches—one running forward into the vertical part of the frontal bone, and one backward into the right middle fossa and through the petrous portion into the foramen lacerum posterius with a subdivision running upward through the squamous portion of the temporal into the parietal bone; thick epidural clot extending from right orbit into middle fossa and over parietal region anteriorly; slight opacity of the arachnoid membrane; hyperæmia of the pia mater; moderate cerebral hyperæmia and œdema and moderate amount of bloody fluid in the lateral ventricles; basal ganglia sodden and flattened; brain convolutions much flattened.

The fluid which primarily escaped from the ear in this last case could only have been the normal cerebrospinal secretion; but the recurrence of the discharge in greater quantity on the second day coincidently with the development of symptoms of inflammation suggested a change from the secretion to the effusion of a subacute arachnitis. This view is strengthened by the post-mortem observations of the flattening of the cerebral convolutions and the sodden appearance of the basal ganglia, and by the absence of the effusion, by which these conditions must have been occasioned.

Extrusion of brain tissue, as a symptom, requires little consideration; that it involves fracture as a necessary precedent is self evident. It occurs in a certain proportion of cases of crushing fractures of the vault, and may then be important as indicating the extent of injury. The loss of large masses of brain matter with recovery of the patient has been often noted. Many instances have been recorded in which with almost entire destruction of the right frontal lobe there has not been even temporary functional impairment. Some of these, as well as a case of recovery after similar destruction of the right cerebrum, were referred to in the first of the two papers upon "Left Frontal Lobe Localization."[3] Two of the original cases which comprised the series published in that connection were also of the same character. One, in which at least two-thirds of the right frontal lobe was destroyed, recovered without serious symptoms, though he became epileptic eight years later; the other, with somewhat less extensive destruction of the left lobe, was temporarily demented, but with ultimate restoration of the mental faculties.

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[3] Phelps: Am. Jour. of Medical Sciences, May–June, 1902.

The escape of brain matter from the ear occurred in three cases, of which two were fatal. In one of the latter it was accompanied by profuse primary aural hemorrhage; in the other it was also primary and accompanied by hemorrhage from the nose and mouth, but by none from the ear. Death ensued in the first on the fourth day, and in the second after a few hours. The recovering case was included in the series collected in the "Treatise upon Intracranial Injuries,"[4] and is again noted among the illustrative cases of the present publication. It occurred on the second day, followed profuse primary hemorrhage from the ear, and continued for twenty-four hours. It is of interest as showing that loss of brain tissue with basal fracture is no more necessarily fatal than fracture of the vault; and also as a direct demonstration that recovery may follow inaccessible cerebral lacerations.

There was extrusion of brain matter through the nose in one case which was fatal; and it also occurred into the orbit in a case of gunshot wound and was discovered in the course of operation for excision of the eye.

Implication of the Cranial Nerves.—This accident like the extrusion of brain matter is of very exceptional occurrence; and before its causative relation to functional loss or disturbance can be admitted, intracranial lesion must be absolutely excluded. It is possible that any cranial nerve may suffer structural injury in its exit from the cranium; but, except in case of the second and seventh pairs, it is in the highest degree improbable, and even with these, as was stated, it is exceptional. Some degree of facial paralysis is among the frequent symptoms of head injuries, and the petrous portion of the temporal bone through which the facial nerve passes is in the line of fracture in a large proportion of basal fractures; yet a lesion of the nerve in its petrous canal has been disclosed upon necropsy in not more than one instance, or at the most two instances, of the whole series.

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[4] *Lib. cit.*, Case CCLXIV.

The compression of the optic nerve by orbital fragments was rather more frequent, but occurred in only six cases, scarcely more than 1 per cent. of the 570 basal fractures. All of these were comprised in the first series of 500 cases.

"Four were recognized only upon necropsy, and of these three had died without recovery of consciousness, and the fourth had suffered no loss of vision. In the two cases in which life was preserved the patient upon restoration of intelligent consciousness discovered that he was blind. Ophthalmoscopic examination in the first case, made on the third day, was negative, though the pupil did not respond to direct exposure to light; fifteen days later atrophy of the optic nerve had begun. In the second case the ophthalmoscopic examination was not made until the fourth week; the pupil was then insensitive to light, and atrophy of the nerve was in progress. Entire loss of vision was permanent in both cases."[5]

If there were other cases of this character they could have been only among those of deaths without restoration of consciousness and without necropsy.

Callan,[6] who has the histories of twenty-five cases, states that "monocular blindness is immediate, and generally with total loss of light perception. The eyeball protrudes and diverges, and the pupil is enlarged and non-responsive to light. Optic nerve atrophy begins within two weeks."

The two cases cited conform to this description except that hemorrhage chanced to be insufficient to cause ocular protrusion or divergence.

Localized pain is not included in the usual category of symptoms of basal fracture. It has been often noted, however, in this series of cases, and its significance often established in subsequent post-mortem examinations. Its intensity and narrow limitation serve to distinguish it from the more diffused frontal or occipital headaches of intracranial injuries, and it is disproportionate to the amount of superficial contusion. It is most likely to be serviceable in the diagnosis of fractures beginning in the occipital fossa, and is sometimes the only direct symptom present. Its seat in such cases may be either in the occipital region or over the mastoid process.

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[5] *Lib. cit.*, p. 25.

[6] Jour. Am. Med. Assoc., March 5, 1892.

How many cases of unrecognized and unsuspected basal fractures there may be is problematical. That there are such is evident from their occasional disclosure post mortem, when none of their enumerated symptoms have existed. It might be reasonable to infer that the more pronounced the indications of intracranial lesion, the greater the probability of the existence of fracture; but it is also to be borne in mind that equally severe and characteristic symptoms of intracranial injury may exist when fracture is absent.

PROGNOSIS.

The results of a study of these cases, so far as it concerns prognosis and treatment, must by reason of limited space be stated in an abbreviated and perhaps somewhat dogmatic form.

Linear fracture, comminuted fracture without loss of substance, and depressed fracture in which the fragments have been replaced in their normal position, unite by definitive callus; and no trace of the osseous lesion remains. Three exceptional cases were observed: one in which a basal fracture through both middle fossæ had united with some displacement of the segments; one which crossed the frontal bone immediately above the orbital ridges and had similarly united with slight displacement; and a third in which an open fissure in the median line of the frontal bone, with appreciable separation and mobility, remained after five years had elapsed.

A loss of osseous substance is replaced by a dense fibrous structure composed of the thickened and consolidated dura mater and periosteum; and is a source of danger in so far as it is a loss of efficient protection from external violence. This danger is commensurate with the extent of osseous deficit and modified by its cranial site. Completely detached fragments separated from periosteum and dura mater, when unremoved, will necrose and may become the medium of infection.

Depressed fragments are confined to the vault and to the orbit, and with early elevation have in general no more serious results than when unattended with displacement. The prognosis may be said to depend largely upon treatment.

The only danger attributable to fracture in itself, whether of the vault or base, is the laceration of brain tissue, or of venous sinuses or other large vessels, by displaced osseous fragments; or the possible implication of an optic or facial nerve in the line of separation.

It is essential to exclude the coincident intracranial injuries in a consideration of the prognosis of fractures. They are not properly complications, as before stated, except in the few instances noted in which consecutive wounds of the brain or greater meningeal vessels have been inflicted by dislocated osseous fragments; but are rather the coexistent results of a common cause—the external violence to which cranium and cranial contents have been alike subjected. It is the failure to recognize this distinction which has occasioned much confusion in both prognosis and treatment.

TREATMENT.

All fractures of the vault raise a question of operation. The treatment here formulated has been the uniform procedure in this series of cases, and is not only rational in theory, but has been justified in practice.

During the continuance of shock no local interference is permissible unless it may be for repression of hemorrhage, or for the aseptic protection of a wound when a fracture is compound. As soon as practicable after reaction has been established, careful examination, and, if necessary to resolve a doubt, thorough exploration should be made of a site of suspected fracture. If no osseous depression or line of fracture can be discovered, and there is no considerable hæmatoma to hamper examination, it may be assumed that no fracture, or at most only a negligible fissure exists. If, however, the existence of fracture is discovered by palpation or if the presence of hæmatoma prevents a positive conclusion, explorative incision even in the absence of indications of intracranial lesion is demanded. It may be that no fracture will be discovered—if so, primary union under aseptic precautions may be accounted certain, and no conceivable possibility of danger will result from a procedure which has made diagnosis beyond question.

A fine closed fissure in the absence of intracranial complication may be neglected; but an open fissure gives sufficient probability of comminution or depression of the inner table to require further exploration by means of chisel, burr drill, or rongeur.

Depressed osseous fragments are to be primarily elevated under all circumstances, after the occurrence of reaction, except in the possible contingency of a laceration of the lateral sinus, when the extraction of a penetrating fragment may be more safely deferred (see Case XXVIII).

Comminuted fractures should be treated as multiple fissures, with especial care given to depressed fragments. Detached pieces of bone should be removed only when no considerable dural or periosteal attachments remain.

Punctured fractures should be explored when attended by local or general symptoms.

The simplest mechanical means should be employed in the exploration and rectification of cranial fractures. The periosteal elevator, reinforced when necessary by the rongeur, chisel, or burr drill, will ordinarily suffice. The trephine will rarely be required, and should be avoided unless indispensable.

Wounds of the scalp even when primarily infected are devoid of danger in the absence of more distant infection, provided aseptic, or if it has become necessary, antiseptic treatment is carefully maintained. Compound fractures thus become no more serious than those which are simple, and explorative incisions require no justification.

The considerations which have determined treatment as here formulated have been sufficiently set forth in earlier publications.[7]

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[7] Phelps: *Lib cit.*, p. 32; N. Y. Medical Journal, June 11, 1902; Annals of Surgery, Dec., 1906.

Direct fractures of the base and the basal continuation of fractures of the vault rarely admit interference. The ethmoid and orbital bones, when subjected to direct violence, may require removal of fragments or the use of aseptic precautions; and fractures of the petrous portion made compound by rupture of the tympanum are at least theoretically made safer by aseptic protection of the auditory meatus. A fracture through the posterior inferior fossa in a single instance was exposed for the relief of hemorrhage (see Case XXVIII).

PATHOLOGY.

The traumatic intracranial lesions may be classified *primarily* as *hemorrhage, contusion,* and *laceration;* and their *sequelæ* as *acute* and *subacute meningeal* and *parenchymatous inflammation*—the latter terminating in *abscess* or *sclerosis.* There is additionally a secondary necrosis of the cerebral tissue independent of inflammation.

The primary conditions may occur as isolated lesions or in combination with each other; and the later inflammations which may also coexist may be developed at any period during the persistence of the direct structural changes upon which they in part depend.

As previously stated, when the intracranial lesions occur as complications of fracture they may be coincident or consecutive and usually dominate the symptomatology, afford the indications for treatment, and determine the prognosis of the case.

HEMORRHAGE.—The intracranial hemorrhages should be classified anatomically as *epidural, pial, cortical,* and *intracerebral.*

Epidural hemorrhage is the only one the origin of which is not exactly indicated by its name. This variation in nomenclature is made necessary by the fact that its source is multiple. It may be derived either from the vessels of the diplöe or of the dura mater, or from the dural sinuses; and may vary in amount from an inconsiderable effusion from the diplöic structures to one from the larger meningeal vessels so great as to immediately compromise the life of the patient.

The usual source of very large extravasations is the arteria meningea media or one of its primitive branches. Rupture of this vessel occurred and was discovered post mortem in twenty-five cases, twenty-one of which were accompanied by fracture through a middle basal fossa. In one the trunk of the artery was traversed by a bullet, and in one was penetrated at its bifurcation by the apex of a small triangular fragment of the inner table of bone. In all the other cases of laceration of this artery with basal fracture, as well as in one with fracture confined to the vertex, and in three without fracture, force was directly exerted upon the vessel. In probably all cases it was extended to the general intracranial contents as indicated by post-mortem appearances. The immediate effect was to strongly compress some considerable portion of a hemisphere, which it would seem should squeeze the blood from the meningeal and cerebral vessels, as has been stated by writers to be really the case; but in fact, with a single exception, these vessels were found in every instance to be more or less congested, which is explicable only by a general intracranial contusion. The danger of compression and the visible area of its extent varies with time and other circumstances which determine the amount of hemorrhage. The lateral and basal aspects of the hemisphere anteriorly are primarily affected, but pressure may extend to the posterior regions. The degree of compression may be sufficient not only to flatten but to make concave the cerebral surface, and may even lift it clear of the basal fossa, so that when the clot is removed the whole fossa, including the petrous portion, may be left empty and exposed, and the brain apparently suspended from the vertex. (See Case LXXVII, First Series, *lib. cit.*)

Hemorrhages of such magnitude and such origin are probably always fatal, unless relief is afforded by operation, and even then recovery is exceptional. The shock of the primary loss of blood, and the added shock of an operation, always difficult on account of the inaccessibility of the lesion, are likely to involve too great a demand upon the recuperative powers of the patient.

The most profuse epidural hemorrhages, however, are not invariably due to rupture of this arterial trunk or its primitive branches. In one case, for instance, a large clot which flattened the whole frontal lobe was derived from a minor meningeal vessel, and in another both occipital lobes were compressed by a hemorrhage from numerous vessels beneath a comminuted fracture. In a third case a whole hemisphere was covered with a thick mass of partially coagulated blood when careful examination showed the larger meningeal vessels to be intact; and in a final instance, one in which a whole hemisphere was compressed to the last degree, the hemorrhage was found to come from some inaccessible point or points in the occipital fossa, and was controlled by gauze pressure between the bone and the dura mater. Operation in such cases as these is oftener successful, since the bleeding vessels are both smaller and more likely to be accessible.

Hemorrhage from wound or rupture of the venous sinuses is usually less profuse, and in case of the superior longitudinal sinus more amenable to treatment. There were eight cases in which this sinus was wounded—seven by a depressed osseous fragment, and one by a sharp instrument. The hemorrhage in each was moderate and readily controlled by plugging or some form of pressure. Recovery followed in six cases. There were four cases of laceration of a basal sinus—two of which were of a lateral, one of a sigmoid, and one of the cavernous sinus. There is no reason to suppose that in such cases operation can be successful. The lateral sinus alone is accessible for interference, the amount of blood necessarily lost is large, and the vicinage of the bulb seems an especial danger.

The two instances of laceration of sinuses not often injured are on account of their infrequency worthy of note.

Case XXVI.—*Laceration of Cavernous Sinus.*—Fracture extended from a point just above supra-orbital notch through orbital plate, lesser sphenoid wing, and groove for cavernous sinus, to foramen lacerum posterius; laceration one-third of an inch long in wall of cavernous sinus; epidural hemorrhage of about two ounces in middle fossa. The patient, who had fallen thirty feet through a ship's hatchway, died from cerebral contusion. There were no ocular paralyses; the corresponding pupil began to dilate two hours after admission to the hospital, and thirty minutes later was widely dilated and insensitive.

CASE XXVII.—*Rupture of Sigmoid Sinus.*—Multiple fractures: linear fracture, eight inches long, just in front of and parallel to coronal suture; depressed fracture one and one-half inches square involving frontal and parietal bones; comminuted fracture at parieto-occipito-squamous junction; and fissures extending from middle, through petrous portion, into posterior fossa; large epidural hemorrhage from rupture of the sigmoid sinus in the line of a fissure. The patient fell three stories to the pavement.

One of the cases of laceration of a lateral sinus is also worthy of special note on account of operation.

CASE XXVIII.—*Laceration of Lateral Sinus: Operation.*—A man fell a distance of from twenty to twenty-five feet, and was at once admitted to the hospital. A few hours later ecchymosis and swelling appeared in the left mastoid and cervical regions, which on the third day had greatly increased. Incision was then made in the inferior occipital region and a comminuted fracture disclosed. Osseous fragments were removed, including one of triangular form. After an appreciable interval profuse hemorrhage followed from some inaccessible point, which was readily controlled by a gauze tampon. Respiration soon began to fail, but was artificially re-established. The pulse for a time remained of good character. Death occurred two hours later.

The triangular fragment was found upon necropsy to have penetrated the lateral sinus, which was lacerated and contused in a space of one-half inch. Epidural hemorrhage, which was confined to the posterior fossæ, was not in great amount. The brain lesions were numerous.

In the great majority of cases an epidural hemorrhage is less exclusively the fatal lesion. It is but one of two or more pathological conditions which contribute in various degrees, according to their relative importance, to the fatal result.

Pial hemorrhage is a result of contusion of the pia mater and the blood when in moderate amount is confined to the meshes of the pial membrane in which it is infiltrated. It then appears in one or several patches of variable size situated in any region of the base or vertex. If in somewhat larger amount it forms a sheet, still confined to the pial meshes, which may cover the entire vertex. If still more profuse it will break either into the subarachnoid cavity or directly upon the cortical surface. A statement previously made that the quantity of blood effused "is never so great as it may be in epidural hemorrhage and that its clinical importance is mainly due to its association with other lesions" (*lib. cit.*, p. 46), requires modification. Such a generalization has proved too broad. The cases in which it is "large enough not only to occasion symptoms of general and local pressure, but to destroy or endanger life," while perhaps comparatively infrequent, are not exceptional. The source of hemorrhage, even when this extravasation is large, is almost always confined to the arterioles and capillaries. Two exceptional instances were noted—one in which nearly the whole hemisphere was flattened by a hemorrhage from a small vein just in front of the first frontal convolution; and a second, included in the "Second Series of Left Frontal Lesions,"[8] in which a hæmatoma extending along the ascending portion of the Sylvian fissure was formed by rupture of the left Sylvian artery. Death in the first instance mentioned seemed to be wholly or essentially the result of hemorrhage. Even when the extravasation is in moderate amount it may of itself occasion death, as occurred in many of the cases of direct compression of the respiratory ganglia.

This hemorrhage has sometimes been improperly called pia-arachnoid. The pia mater is unquestionably a subserous connective tissue analogous to that of the pleura and the peritoneum, and like that serves for the subdivision of the nutrient vessels of the superjacent serous membrane. It differs from other subserous layers, however, in this, that it also serves for the subdivision of the nutrient vessels not only of the cortex but of the whole substance of the viscus which it covers. This function is so important that, unlike the other subserous tissues, it has received a distinctive name; and while, as pointed out by Dr. Alonzo Clark many years ago, the inflammation of which the processes are largely manifested in its substance is an arachnitis, there is no reason why its hemorrhages or œdemas, with which the arachnoid membrane has no connection, should be termed otherwise than pial. To combine in name the two membranes, as pia-arachnoid, ignores the function of the pia mater; to call the hemorrhage into the pial tissue pia-arachnoid assumes a relation which does not exist.

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[8] Phelps: Am. Jour. Med. Sci., March, 1906, Case XI.

Cortical hemorrhage is occasioned by a laceration of the brain substance, and is primarily situated upon the cerebral cortex, and beneath the pia mater. It is often profuse and then breaks through the pia mater into the subarachnoid space, and, as in one case in which the dura mater had been wounded, may even spread over the epidural surface. The cerebral wound may be cortical, or the hemorrhage from a subcortical laceration may reach the surface by rupture of intervening tissues. The extravasation is sometimes slight, but is more frequently excessive than when pial or epidural; and as lacerations are most frequent at the cerebral base, it especially affects the basal fossæ.

These several forms of hemorrhage often coexist, and the pial and cortical are not infrequently commingled. They are even then usually capable of discrimination. The epidural hemorrhage is at once recognized by its topographical relations to the dura mater; the cortical hemorrhage can always be traced to a wound of the cerebral surface; and the pial hemorrhage when uncomplicated by the cortical is differentiated by exclusion—by its situation beneath the dura mater, and by the absence of cortical wound or rupture. The pial and cortical hemorrhages when coexistent are often situated in different cerebral regions; but when a cortical extravasation breaks into the pia mater it is of course impossible to determine whether a pial hemorrhage had pre-existed in the same area.

Intracerebral hemorrhage is so related to subcortical laceration, either as cause or effect, that it will be best to consider it in that connection.

The different intracranial hemorrhages occur with about equal frequency, and one or more of them are present in the vast majority of cases. Their pathic effects vary in accordance with their extent and relation to other lesions rather than with their source.

It seems probable that neither form of hemorrhage is ever an isolated lesion. The same violence which is sufficient to rupture a dural or pial vessel, or to produce a superficial cerebral wound, can hardly fail to be further transmitted and diffused through the whole or some considerable portion of the brain substance; that general circulatory changes have occurred is an obvious fact in a very large proportion of necropsic examinations, in which obvious facts are so often overlooked. It becomes a question therefore what symptomatic conditions may be due to hemorrhage and what to concurrent lesions; and a still further question, in what manner hemorrhage produces the effects for which it is properly responsible. There are cases of death as to the causes of which there can be no question—as in shock, exsanguination, or compression of the respiratory ganglia. The greater number of deaths, those which occur when hemorrhage seems to have been an essential lesion, can be less simply interpreted. It has been taught that a hemorrhage, or even an osseous fragment of inconsiderable size, by mechanical compression of the cerebral vessels and their exsanguination, produces a cerebral anæmia which affords adequate explanation for consecutive symptoms; and yet the osseous fragment may be of size quite insufficient to appreciably reduce the cranial contents, or the hemorrhage may be attended by a general cerebral hyperæmia, and not by anæmia. It has been proven by experimentation that mechanical compression induced without violence may cause fatal coma through a progressive cerebral anæmia; and it may be admitted that an intracranial hemorrhage under certain conditions may cause death in the same manner. It still leaves for explanation the greater number of cases—the typical cases—in which minor hemorrhages with similar symptoms have been followed or attended by an opposite circulatory condition.

It has been customary to link together all encroachments upon the intracranial space under one common name, compression, and to assume common structural changes indicated by certain identical symptoms. In fact, their one common characteristic is of no material importance; their pathology is essentially diverse. The apparent results of osseous depressions and of the majority of hemorrhages depend upon concurrent lesions; and the mechanical effect of inflammatory products is lost in the manifestations of the primary disorder which produces them. The local pressure which results from minor encroachments upon cranial capacity differs from compression which is general, and it is only when it is exerted upon cerebral centres of functional activity that it has indications of its own.

Hemorrhages of moderate extent are probably removed in cases of recovery by direct absorption; and this removal is doubtless facilitated in those cases in which some portion has been previously removed by operation. Large clots may be encysted and liquefied prior to absorption, and such have been observed post mortem in various stages of this process. The cysts may become permanent and are often discovered beneath unrelieved depressed fractures of long standing in the course of operations for the relief of epilepsy.

(To be continued.)